DB Name	Query	Hit Count Set Name	
PGPB	((her-2) or her2 or (c-erb-B2) or (erb-B2) or (c-erb-b-2))	5	<u>L17</u>
JPAB,EPAB,DWPI	115 and 113	1 .	<u>L16</u>
JPAB,EPAB,DWPI	t near cell near epitope\$1	362	<u>L15</u>
JPAB,EPAB,DWPI	112 and 113	0	<u>L14</u>
JPAB,EPAB,DWPI	((her-2) or her2 or (c-erb-B2) or (erb-B2) or (c-erb-b-2))	87	<u>L13</u>
JPAB,EPAB,DWPI	\$3apeptide	926	<u>L12</u>
USPT	19 same 13	0	<u>L11</u>
USPT	19 and 13	13	<u>L10</u>
USPT	\$3apeptide	3876	<u>L9</u>
USPT	\$peptide	52872	<u>L8</u>
USPT	15 same 13	- 1	- · <u>L7</u>
USPT	15 and 13	34	<u>L6</u>
USPT	t near cell near epitope\$1	708	<u>L5</u>
USPT	11 and 13		<u>L4</u>
USPT	((her-2) or her2 or (c-erb-B2) or (erb-B2) or (c-erb-b-2))	516	<u>L3</u>
USPT	11 and ((her-2) or her2 or (c-erb-B2) or (erb-B2) or (c-erb-b-2))	0	<u>L2</u>
USPT	nonapeptides	248	<u>L1</u>

WEST

Generate Collection

L17: Entry 3 of 5

File: PGPB

Jul 5, 2001

PGPUB-DOCUMENT-NUMBER: 20010007152 PGPUB-FILING-TYPE: new-utility

DOCUMENT-IDENTIFIER: US 20010007152 A1

TITLE: RECOMBINANT CONSTRUCTS ENCODING T CELL RECEPTORS SPECIFIC

FOR HUMAN HLA-RESTRICTED TUMOR ANTIGENS

PUBLICATION-DATE: July 5, 2001

INVENTOR-INFORMATION:

NAME CITY STATE COUNTRY RULE-47

SHERMAN, LINDA A. LA JOLLA CA US

LUSTGARTEN, JOSEPH LA JOLLA CA US

APPL-NO: 08/ 812393

DATE FILED: March 5, 1997

CONTINUED PROSECUTION APPLICATION: CPA

RELATED-US-APPL-DATA:

RLAN RLFD RLPC RLKC RLAC

60012845 Mar 5, 1996 US

INT-CL: [07] A01K 67/027

US-CL-PUBLISHED: 800/4; 800/21, 435/91.1, 435/91.2 US-CL-CURRENT: 800/4; 435/91.1, 435/91.2, 800/21

REPRESENTATIVE-FIGURE: NONE

ABSTRACT:

Methods are described to obtain nucleic acid molecules that encode T cell receptors and their derivatives that are human HLA-restricted and which are specific for tumor-associated antigens found in human tumors. These nucleic acids are useful in preparing recombinant cells for diagnosis and therapy of human tumors.

ANSWER 7 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: DOCUMENT NUMBER:

1995:111204 BIOSIS PREV199598125504

TITLE:

Sequence motifs of human HER-2

proto-oncogene important for peptide binding to HLA-A2.

AUTHOR(S):

Fisk, Bryan; Chesak, Brad; Ioannides, Maria G.; Wharton,

Taylor; Ioannides, Constantin G. (1)

CORPORATE SOURCE:

(1) Dep. Gynecol. Oncol., 1515 Holcombe Boulevard, Box 67,

Houston, TX 77030 USA

SOURCE:

International Journal of Oncology, (1994) Vol. 5, No. 1,

pp. 51-63.

ISSN: 1019-6439.

DOCUMENT TYPE:

Article

LANGUAGE:

English

Tumor progression and metastasis are often associated with overexpression of specific cellular proteins. In 1991, we introduced a hypothesis that epitopes of nonmutated overexpressed proteins can be targets of a

cellular immune response against tumor mediated by T cells (Mol

Carcinogen

6: 77-81, 1992) and that, when T cell epitopes are present, distinction between tumor immunity/autoimmunity and unresponsiveness can be

on the protein concentration as a limiting factor of epitope supply. In support of this hypothesis. we demonstrated that CTL from patients with ovarian tumors which overexpress HER-2 protooncogene can recognize both autologous tumor and synthetic analogs of a specific epitope from HER-2, which was identified based on the convergence of all criteria for selection of HLA-A2 associated epitopes recognized by T cells. In this study, we identified all epitopes in HER-2 containing nonapeptides with HLA-A2

anchors. Of these, analysis of potential amphiphilic sites identified

both

sequences and specific mutations that positively affected the reactivity of conformationally dependent HLA-A2 specific mAb which served as an indication of HER-2 peptide binding. We also report the in vitro induction of cellular responses to these peptides by PBMC from healthy HLA-A2+ volunteers as an indication of their ability to stimulate/restimulate preexisting T cell responses to HER-2. The peptides induced proliferative responses in one of four donors tested and CTL responses (one of three peptides tested in two of three donors). This strategy may allow selection of immunogenic HER-2 peptides and elucidation of mechanisms operating in induction of tolerance to defined epitopes on self-proteins.

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